Emotional Reactivity, Emotion Regulation Capacity, and Posttraumatic Stress Disorder in Traumatized Refugees: An Experimental Investigation

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Abstract: Refugees who suffer from posttraumatic stress disorder (PTSD) often react with strong emotions when confronted with trauma reminders. In this study, we aimed to investigate the associations between low emotion regulation capacity (as indexed by low heart rate variability [HRV]), probable PTSD diagnosis, and fear and anger reaction and recovery to trauma-related stimuli. Participants were 81 trauma-exposed refugees (probable PTSD, n = 23; trauma-exposed controls, n = 58). The experiment comprised three 5-min phases: a resting phase (baseline); an exposition phase, during which participants were exposed to trauma-related images (stimulus); and another resting phase (recovery). We assessed HRV at baseline, and fear and anger were rated at the end of each phase. Linear mixed model analyses were used to investigate the associations between baseline HRV and probable DSM-5 PTSD diagnosis in influencing anger and fear responses both immediately after viewing trauma-related stimuli and at the end of the recovery phase. Compared to controls, participants with probable PTSD showed a greater increase in fear from baseline to stimulus presentation, d = 0.606. Compared to participants with low emotion regulation capacity, participants with high emotion regulation capacity showed a smaller reduction in anger from stimulus presentation to recovery, d = 0.548. Our findings indicated that following exposure to trauma-related stimuli, probable PTSD diagnosis predicted increased fear reactivity, and low emotion regulation capacity predicted decreased anger recovery. Impaired anger recovery following trauma reminders in the context of low emotion regulation capacity might contribute to the increased levels of anger found in postconflict samples.

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Tobias R. Spiller

University of Zurich

Belinda J. Liddell

University of New South Wales

Matthis Schick

University of Zurich

Naser Morina

University of Zurich

Ulrich Schnyder

University of Zurich

Monique Pfaltz

University of Zurich

Richard A. Bryant

University of New South Wales

Angela Nickerson

University of New South Wales

Author Note

Tobias R. Spiller, University of Zurich, University Hospital Zurich, Department for Psychiatry; Belinda J. Liddell, School of Psychology, University of New South Wales; Matthis Schick, University of Zurich, University Hospital Zurich, Department for Psychiatry; Naser Morina, University of Zurich, University Hospital Zurich, Department for Psychiatry; Ulrich Schnyder, University of Zurich, University Hospital Zurich, Department for Psychiatry; Monique Pfaltz, University of Zurich, University Hospital Zurich, Department for Psychiatry; Richard A. Bryant, School of Psychology, University of New South Wales and Angela Nickerson, School of Psychology, University of New South Wales.

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Correspondence concerning this article should be addressed to Angela Nickerson, PhD, School of Psychology, UNSW Australia, Sydney, NSW 2052, Australia. E-Mail: a.nickerson@unsw.edu.au

Abstract

Refugees suffering from posttraumatic stress disorder (PTSD) often react with strong emotions when confronted with trauma reminders. This study aimed to investigate the associations between low emotion regulation capacity, as indexed by low heart rate variability (HRV), probable PTSD diagnosis and fear and anger reaction and recovery to trauma-related stimuli. Participants were 81 trauma-exposed refugees (probable PTSD = 23; trauma-exposed controls = 58). The experiment comprised three phases, each lasting five minutes: a resting phase (baseline), an exposition phase, during which participants were exposed to trauma-related images (stimulus), and another resting phase (recovery). HRV was assessed at baseline, and fear and anger were rated at the end of each phase. We used linear mixed model analyses to investigate the associations between baseline HRV and probable DSM-5 PTSD diagnosis in influencing anger and fear responses both immediately after viewing trauma-related stimuli, and at the end of the recovery phase. Compared to controls, participants with probable PTSD showed a greater increase in fear from baseline to stimulus presentation (d = 0.606). Compared to participants with high emotion regulation capacity, participants with low emotion regulation capacity showed a smaller reduction in anger from stimulus presentation to recovery (d = 0.548). Our findings indicate that probable PTSD diagnosis predicted increased fear reactivity and low emotion regulation capacity predicted decreased anger recovery following exposure to trauma-related stimuli. Impaired anger recovery following trauma reminders in the context of low emotion regulation capacity might contribute to the increased levels of anger found in post-conflict samples.

Keywords: PTSD, refugees, heart rate variability, anger, fear, emotion regulation

By definition, refugees and asylum-seekers experience high levels of exposure to potentially traumatic events (PTEs), including human rights violations. A dose-effect relationship (Mollica, McInnes, Poole, & Tor, 1998) between PTE exposure and risk for mental disorders such as posttraumatic stress disorder (PTSD) and depression is well established, with the prolonged, repeated and human-instigated nature of the traumatic events having far-reaching mental health consequences (Silove et al., 2014). Further, even after individuals reach a situation of relative safety (e.g., a resettlement country), many refugees and asylum-seekers continue to be exposed to trauma reminders in the form of news from the home country or contacts with family members and friends who live in circumstances of persecution and conflict. Despite this, relatively little is known about the emotional responses of refugees to trauma reminders, nor how their capacity to manage their emotions influences these reactions.

While little research has investigated emotional responses to trauma reminders in refugees, there has been relatively more study of this phenomenon in non-refugee trauma survivors (e.g. Ehlers, Hackmann, & Michael, 2004). Much research has investigated the role of fear in influencing the mental health of trauma survivors, with current dominant biological models of PTSD drawing from fear learning principles (e.g. Etkin & Wager, 2007). In addition, the role of anger in PTSD has also been a topic of major interest, in particular because explosive anger reactions have been negatively related to treatment outcome (Forbes et al., 2008), violent behavior (Blakey, Love, Lindquist, Beckham, & Elbogen, 2017) and intimate relationship problems (Taft, Watkins, Stafford, Street, & Monson, 2011). Most experimental studies investigating responses to trauma-related stimuli have focused on changes in psychophysiological (e.g. Pitman, Orr, Forgue, de Jong, & Claiborn, 1987) or brain imaging measures upon encountering trauma reminders (e.g. Shin et al., 1999).

Only four studies have assessed discrete affective response to trauma-related stimuli (Amdur, Larsen, & Liberzon, 2000; Pitman et al., 1987; Shin et al., 1999, 2004). These four studies used different samples (e.g. survivors of childhood sexual abuse or combat veterans), different control groups (trauma-naïve or trauma-exposed), different stimuli (script-driven imagery procedure or visual stimuli) and reported conflicting results regarding the affective response to trauma-related stimuli. Two studies of these four studies found an increased fear reaction in PTSD sufferers (Pitman et al., 1987; Shin et al., 2004) and two did not (Amdur et al., 2000; Shin et al., 1999). Similarly, two studies reported increased anger after stimulus exposure in participants suffering from PTSD (Amdur et al., 2000; Pitman et al., 1987) and also two did not (Shin et al., 1999, 2004).

Alongside the research interest in affective responses to trauma reminders, there has been growing research attention on the relationship between emotion regulation and PTSD, with individuals with PTSD consistently demonstrating poorer emotion regulation capacities compared to trauma-exposed controls (Ehring & Quack, 2010). Few studies have investigated the relationship between emotion regulation capacity and emotional reactivity to aversive stimuli. Badour and Feldner (2013) found that emotional reactivity to script-driven imagery was influenced by the interaction between PTSD symptoms and emotion regulation capacity; individuals with more severe PTSD symptoms and diminished emotion regulation capacity reported increased fear responses to trauma cues. In a refugee samples, Nickerson and colleagues (2015) found that torture survivors who regularly used emotional suppression (a maladaptive emotion regulation strategy) experienced higher levels of distress when exposed to trauma-related images, while the opposite pattern was observed in non-tortured refugees. In a second study Nickerson and colleagues (2017) found that refugees who were instructed to use an adaptive emotion regulation strategy, and had low levels of trait suppression experienced lower negative affect after exposure to trauma-related stimuli. In addition to only

measuring general negative affect rather than considering discrete emotional responses, a limitation of these studies, was the reliance of self-report measures to assess habitual use of emotion regulation strategies. Working with groups from varied cultural and language backgrounds limits the use of self-report measures to index complex constructs such as emotion regulation. An alternative method of assessing low emotion regulation capacity that is independent of culture and language is parasympathetically mediated heart rate variability (HRV; Appelhans & Luecken, 2006). According to Thayer and Lane (2000) emotion regulation capacities are diminished when the individual is in a state of distress, resulting in a decreased parasympathetic inhibition of heart rate and dominance of the sympathetic nervous system (see also Lane et al., 2009). Consistent with this model, high resting parasympathetically mediated HRV indicates a system with high adaptive potential to the environment, or in terms of emotion, a system with strong emotion regulation capacity (Appelhans & Luecken, 2006; Williams et al., 2015). Over the last 10 years, research has demonstrated that low resting HRV is associated with mental disorders, including PTSD (Nagpal, Gleichauf, & Ginsberg, 2013; Zoladz & Diamond, 2013), suggesting diminished emotion regulation capacity in these populations. In addition, low HRV has been theorized to be a predictive biomarker of risk for developing PTSD (Shah & Vaccarino, 2015).These findings have been extended to post-conflict groups in a study conducted by Liddell and colleagues (2016) in Timor-Leste, which found that the combination of increased PTSD symptoms and reduced age-related HRV partly mediated the relationship between PTE exposure and psychological distress and aggression. Taken together, this research suggests that HRV is a potentially valuable indicator of a valid indicator of emotion regulation capacity in trauma survivors across cultural groups.

The goals of the current study were to (1) investigate fear and anger responses and recovery following trauma-related stimuli are different in refugees, (2) examine the

association between probable PTSD diagnosis and emotional reactivity/recovery, and (3) investigate the association between high vs low emotion regulation capacity (as indexed by HRV) and emotional reactivity/recovery. To date, there are conflicting experimental results relating to the impact of PTSD on fear and anger reactivity following exposure to trauma-related stimuli (Amdur et al., 2000; Pitman et al., 1987; Shin et al., 1999, 2004). However, increased fear reaction is by definition a core component of PTSD, and there are emerging findings showing heightened levels of explosive anger in post-conflict populations (Silove et al., 2009). Therefore, we predicted first that refugees with a probable diagnosis of PTSD would evidence increased fear and anger reactions and impaired emotional recovery when exposed to trauma-related stimuli compared to refugees without PTSD. Second, we hypothesized that refugees with low emotion regulation capacity (low resting HRV) would experience greater fear and anger responses to trauma-related stimuli and an impaired recovery after stimulus presentation, compared to those with high emotion regulation capacity (high resting HRV). Finally, the associations between emotional response and recovery and the interaction of HRV status and probable PTSD diagnosis were investigated exploratorily.

Method

Participants

Participants were 82 refugees and asylum-seekers, recruited via advertisements at casework and counseling services and community organizations in [edited for review]. Inclusion criteria were (a) 18 years of age or older, and (b) refugee or asylum-seeking status. Due to an incomplete assessment of both PTSD symptoms and resting HRV, one participant was excluded from further analysis, resulting in a final sample size of 81. The mean age of participants was 33.9 years (SD = 9.38), and 66.7 % (n = 54) of the sample were male.

Participants came from a variety of countries-of-origin, including Iran (n = 45, 55.5%), Afghanistan (n = 10, 12.3%), Sri Lanka (n = 8, 9.9%), Iraq (n = 7, 8.6%), and others such as Serbia, Burma and Bhutan (n = 11, 13.6%). Participants had been exposed (experienced or directly witnessed) to a mean of 8.60 (SD = 3.46) types of traumatic events. Exposure to "Lack of food or water" (n = 63, 77.78%), "Serious injury" (n = 63, 77.78%), and "Being close to death" (n = 63, 77.78%) was most common. Exposure to "Rape or sexual abuse" was least frequently reported (n = 10, 12.35%; see Table S1 for details). Participants had been in [edited for review] for a mean of 2.51 years (SD = 3.60) with the majority of the sample held temporary visas or were asylum-seekers (n = 68, 84.0%). A minority had a permanent visa status or were [edited for review] citizens (n = 13, 16.0%). A majority of the participants was not taking any medication (n = 46, 56.1%) at the time of the experiment. Still, a substantial minority (n = 28, 34.1%) was taking medication and for few participants (n = 8, 9.8%) no data on medication use was available.

Procedure

This study was approved by the Human Research Ethics Committee [edited for review]. The experimental sessions were conducted by clinical psychologists with masters or doctoral-level qualifications, with experienced health interpreters if necessary. All measures were administered verbally. In the first session, the purpose of the study was explained to all participants and written consent was obtained. Next, the Harvard Trauma Questionnaire and the PTSD Symptom Scale were administered via interview. Finally, participants were instructed to abstain from alcohol, coffee and smoking 24 hours prior to the experimental session. At the beginning of the second experimental session (one week later), the Polar heart rate monitor was attached to participants. To index resting heart rate, participants were first subjected to a brief habituation phase (one minute) before engaging in a five-minute rest period whilst seated. Next, participants rated their baseline fear and anger on a visual scale

(Baseline Phase). Following this, participants were exposed to 25 trauma-related images presented pseudorandomly on a computer screen for 10 seconds each. Prior to each image, a white cross-centered on a black screen was presented for two seconds. The entire task lasted for five minutes. Thereafter, participants again rated fear and anger on a visual scale (Stimulus Phase). Finally, another five-minute rest period was completed whilst seated. Participants also completed a final rating of fear and anger on a visual scale (Recovery Phase). Participants were reimbursed with $AUD25 for each of the research sessions, which lasted approximately 90 minutes each.

Measures

Trauma Exposure. Exposure to potentially traumatic events was measured using the Harvard Trauma Questionnaire (HTQ; Mollica et al., 1992), which indexes 16 types of traumatic events commonly experienced by refugees. The HTQ yields a total count of types of traumatic events to which an individual has been exposed, ranging from 0 to 16.

Probable PTSD Diagnosis. Probable PTSD diagnosis was assessed in a semi-structured interview using the interview version of the PTSD Symptom Scale (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993). The PSS-I contains seventeen items assessing DSM-IV symptoms of PTSD on a 4-point scale (0 = *not at all*, 3 = *5 or more times per week/very much*). To account for the new symptom criteria included in the DSM-5, four additional items were added (blame of self and/or others; reckless/self-destructive behavior; negative beliefs about the self, others and the world and persistent negative emotional state). Probable PTSD diagnosis was determined by a trained clinician using the above described version of DSM-5 criteria (American Psychiatric Association, 2013), with responses of "2-4 times per week/somewhat" (2) and "5 or more times per week/very much" (3) on each of the items indicating that a specific symptom was present. The PSS-I has previously been used in

research in psychotraumatology with groups from various cultural backgrounds (i.e. Pfeiffer & Elbert, 2011) and the reliability of the total PTSD symptom scale derived from the PSS-I was high (Cronbach's $\alpha = .93$).

Fear and Anger. Fear and anger responses were measured using a five-point Likert scale (1 = not at all [afraid/angry], 5 = extremely [afraid/angry]). Participants were asked at three time-points during the experimental session (prior to and immediately after viewing the images and after a five-minute recovery period) to verbally rate their fear and anger respectively with a full number between 1 and 5.

Trauma-related images. The 25 trauma-related images used in this study depicted scenes of interpersonal violence, civil unrest and natural disasters. These pictures were obtained from the International Affective Picture System (Lang, Bradley, & Cuthbert, 1997), and have been previously demonstrated to elicit an emotional response in refugees (e.g. Spahic-Mihajlovic, Crayton, & Neafsey, 2005)

Heart Rate Variability. A polar RS800CX portable heart rate monitor (Polar Electro Oy, Kempele, Finland) was used to record heart rate with a sampling rate of 1000 Hz. The reliability and validity of recordings from portable heart rate monitors compared with standard electrocardiography (ECG) lab-based measures has been demonstrated previously (Barbosa, Silva, Azevedo, Pastre, & Vanderlei, 2016; Weippert et al., 2010) Kubios HRV (v.2.2, 2014, http://kubios.uef.fi/) was used to process and analyze data. Artefact correction was conducted by applying a default "medium" artefact correction filter first, followed by a higher level of correction filter if necessary by manual inspection. If adequate correction of visually inspected artefacts failed, trimming of the sample was completed to remove the problematic section. Heart rate (beats per minute; bpm) was computed as average number of R-waves per minute using the record of heart rate during the five-minute baseline period.

Fourier transformation was used to analyze Power spectral density of HR, following published standards (Task Force of the European Society of Cardiology, 1996). We chose RMSSD as an index for parasympathetic influences on heart rate, being in line with guidelines for assessment of parasympathetic influences on heart rate over short time periods (i.e. 5 minutes, Task Force of the European Society of Cardiology, 1996). For 5 participants, technical difficulties precluded the collection of the resting HRV data. These participants were therefore excluded from HRV-related analyses, resulting in a final sample size of 76 for these analyses.

Data Analysis

This study aimed to investigate how the affective response and recovery following trauma-related stimuli differs between trauma survivors (a) suffering from probable PTSD or not and (b) with high or low emotion regulation capacity (indexed by high or low resting HRV respectively). Therefore, to study the relationship between probable PTSD diagnosis and subsequent affective response, probable PTSD was determined by trained clinicians (see "Study Measures") and afterwards the sample was split into a "Probable PTSD diagnosis" (n = 23) and a "no PTSD diagnosis" group (n = 58).

This study aimed to map how at risk-groups of traumatized refugees responded to an emotionally provocative task. Risk was categorized in psychological risk (i.e. PTSD diagnosis) and physiological risk (i.e. low HRV). To explore the influence of these two risk factors and their interaction on affective response in a direct manner, we used categorical variables. The categorical approach allowed to explore the intersection between these two risk factors in a more direct manner, then if continuous indices of HRV and PTSD had been used. There are studies showing that baseline HRV is lower in individuals suffering from PTSD compared to controls (Nagpal, Gleichauf, & Ginsberg, 2013). However, to our

knowledge, no absolute cut-offs for defining low or high HRV exist. Therefore, we used a median split to create a low and a high HRV group. Moreover, this approach had the methodological benefit of reducing the influence of age and medication use as potential confounding variables on HRV. Additionally, the chosen method simplified the interpretation of the results, and followed the procedure in other studies investigating the association of low and high resting HRV in PTSD (e.g. Wendt, Neubert, Koenig, Thayer, & Hamm, 2015) or as a measure for emotion regulation capacity (e.g. Williams et al., 2015). We therefore divided the sample into "High HRV" (n = 38) and "Low HRV" (n = 38), using a median split of the resting HRV (RMSSD, median = 26.5 ms). All analyses were conducted using SPSS Version 23 (IBM Corp, Released 2014).

Independent samples t-tests were first used to investigate differences between groups (Low HRV vs. High HRV, Probable PTSD vs. no PTSD) on baseline demographic characteristics. Next, we used linear mixed models to investigate change in fear and anger and their association with resting HRV and probable PTSD diagnosis and their interaction by investigating change of the outcome variable (fear or anger) over time using two models (baseline to stimulus and stimulus to recovery) for each condition (HRV status and probable PTSD diagnosis, and their interaction), resulting in a total of 12 linear mixed models. For all these models, the Level 1 predictor was time-point (namely, including (a) baseline and stimulus phase and (b)stimulus and recovery phase). Random effects in all models included the model intercept and time. Significance level was set to alpha = 0.05 for all analyses.

The first set of analyses investigated the relationship between resting HRV and changes in fear and anger response from baseline to stimulus presentation and from stimulus presentation to recovery. Fixed effects in this model were Time (coded as 0 = baseline, 1 = stimulus phase, 2 = recovery phase), HRV status (coded as 0 = low HRV, 1 = high HRV) and HRV x time interaction. This allowed us to investigate (1) whether fear and anger responses

varied over time over the entire sample, (2) whether fear and anger responses differed overall for those high and low in HRV, and (3) whether the pattern of fear and anger responses differed over time for those high and low in HRV.

The relationship between probable PTSD diagnosis and changes in emotional response and recovery was investigated in the second set of analyses using the same model as stated above but replacing HRV status with probable PTSD diagnosis (coded as 0 = no probable PTSD diagnosis, 1 = probable PTSD diagnosis).

Accordingly, a third set of analyses was used to investigate the relationship between the interaction of HRV and probable PTSD diagnosis and changes in emotional response and recovery. The interaction of HRV status and probable PTSD diagnosis were coded as 0 = Low HRV & PTSD, 1 = Low HRV & PTSD, 2 = High HRV & PTSD, and 3 = High HRV & no PTSD, with Low HRV & PTSD being the reference group for this set of analyses.

One of the above mentioned five participants with missing data from the HRV recording, also had incomplete information regarding probable PTSD diagnosis. Therefore, we excluded these participants for all analyses. There was no data was missing for the remaining participants.

Effect sizes were approximated for all significant findings by calculating Cohen's d of the difference of the change of the dependent variable between the two time points of interest. Effect size calculations were carried out by using JASP Version 0.8.6 (JASP Team, 2018).

Results

Group differences

Group characteristics are presented in Table 1. Independent sample t-tests and chi-squared tests revealed that the low HRV group did not differ from the high HRV group

regarding age, gender, probable PTSD diagnosis, duration of stay [edited for review], medication use and fear and anger at baseline.

Overall affective response and recovery to trauma-related stimuli

Linear mixed models showed that the change of overall fear and anger in all models was significant for Time (for details see Table S2). This indicated that fear and anger increased from baseline to stimulus presentation and decreased from stimulus presentation to recovery period.

Associations between HRV status, and fear and anger response and recovery

Findings are presented in Table 2 and Figure 1. HRV status alone did not significantly predict fear or anger at any time-point. A significant interaction for time x HRV was found in the anger model from stimulus presentation to recovery (with an effect size of Cohen's d = 0.548). This suggests that the low and the high HRV group showed a similar increase in anger from baseline to stimulus phase, but that compared to the high HRV group, the low HRV group showed a smaller decrease in anger ratings. This indicates that both groups reacted with a similar increase in anger to the stimulus exposure, but that the low HRV group had an impaired anger recovery during the resting period following the exposure. There were no group differences in the reaction pattern of fear.

Associations between probable PTSD diagnosis, and fear and anger response and recovery

Findings are presented in Table 3 and Figure 2. Probable PTSD diagnosis alone did not significantly predict fear or anger at any time-point. However, time x probable PTSD diagnosis interaction was significant in the fear model from baseline to stimulus presentation (with an effect size of Cohen's d = 0.606). This suggests that the probable PTSD diagnosis group showed a greater increase in fear from baseline to stimulus presentation compared to

the no PTSD reference group, but that the groups did not differ in fear responses from stimulus to recovery. There were no group differences for anger.

Associations between the interaction of HRV status and probable PTSD diagnosis, and fear and anger response and recovery

Findings are presented in Table S3. The interaction of HRV status and probable PTSD diagnosis alone did not significantly predict fear or anger at any time-point. However, time x interaction of HRV status and probable PTSD diagnosis was significant in the fear model from baseline to stimulus presentation, with both groups not fulfilling PTSD criteria had a decreased fear response when compared to the reference group ("Low HRV & PTSD"), whereas no difference was found between the reference and the "High HRV & PTSD" group. This indicates that increased fear response in PTSD sufferers was independent of HRV status. With regard to the anger model from stimulus presentation to recovery, the only difference was found between the reference group ("Low HRV & PTSD") and the "High HRV & no PTSD" group. This indicates that anger recovery is influenced by both HRV status and PTSD diagnosis and that only the combination of low HRV and PTSD diagnosis led to a decreased anger recovery following stimulus presentation. However, due to the small size of the subgroups (with n = 9 for the smallest) these results were not the main focus of our investigation and should be interpreted with care.

Discussion

The current study represents the first investigation of the influence of emotion regulation capacity, as indexed by resting HRV, and probable PTSD diagnosis on discrete affective responses to trauma-related stimuli in highly traumatized refugees. We found partial support for our first hypothesis. Specifically, refugees with a probable diagnosis of PTSD showed increased fear, but not anger, reactions to trauma-related stimuli. However, this

increased fear reactivity did not extend to the recovery phase. Regarding our second hypothesis, we found that individuals with low resting HRV showed impaired anger recovery, but not anger reactivity in response to aversive cues. These findings suggest that increases in fear reactivity are specifically associated with probable PTSD, but that impaired anger recovery following trauma reminders is associated with low emotion regulation capacity (measured by HRV at rest).

Our finding that anger recovery following exposure to trauma-related stimuli is impaired in trauma survivors with low emotion regulation capacity is novel. These results are consistent with a recent study reporting that the relationship between PTSD and impulsive aggression was fully mediated by self-reported emotion regulation difficulties in combat veterans (Miles, Menefee, Wanner, Teten Tharp, & Kent, 2016) This is also in accordance with the findings from Liddell and colleagues (2016), who reported that the interaction of elevated PTSD symptoms and age-related reduction of HRV partly mediated the relationship between exposure to PTE and aggression in a post-conflict sample. From a clinical perspective, these results indicate that while many trauma survivors, independent of probable PTSD diagnosis, react with anger to trauma-related stimuli, those with high emotion regulation capacity may be better able to cope with this emotion in a short period of time. Those with a low emotion regulation capacity on the other hand, may struggle to control their anger reactions, and show impaired recovery. A potential, but fully speculative, explanation for this might be that anger responses may induce ruminative reactions that persist beyond the presentation of anger-eliciting stimuli, with this process being mainly dependent on one's emotion regulating capacity, rather than probable PTSD diagnosis. Accordingly, research has indicated that the relationship between anger and PTSD is mediated by rumination (Orth, Cahill, Foa, & Maercker, 2008) and that angry rumination following recalling an anger eliciting memory led to higher self-reported anger than other emotion regulating techniques

(Fabiansson, Denson, Moulds, Grisham, & Schira, 2012). These findings may provide a potential explanation for the increased levels of anger in trauma survivors often found on a population level (Foa, Riggs, Massie, & Yarczower, 1995; Hinton, Rasmussen, Nou, Pollack, & Good, 2009). However, this only explains, on a speculative level, why low emotion regulation capacitates might lead to a prolonged anger recovery and falls short to explain why the fear recovery pattern is not affected similarly. Therefore, further investigations are needed to explore and understand the mechanisms underlying the different reactions and recovery patters of fear and anger and whether our findings are specific to refugee populations, who are commonly exposed to very high levels of interpersonal and extreme traumas, or if this generalizes to other populations as well.

Our finding that initial fear responding was elevated in refugees with PTSD indicates that having a probable PTSD diagnosis is particularly associated with increased fear reactivity, but not recovery. These results are in accordance with findings that probable PTSD diagnosis predicts increased response of fear, guilt, and disgust but not anger immediately following an imaginal script-driven trauma exposure (Shin et al., 2004). Partly in line with our findings, (Pitman et al., 1987) found that fear, sadness, and disgust (but also anger) were increased 30 seconds after exposure to a imagery script procedure in individuals with PTSD compared to a combat-exposed control group. Overall, findings from this and previous research (Shin et al., 2004) corroborate major models of PTSD which consider disrupted fear processing and learning to be a central component of the disorder (for review see: Shin & Liberzon, 2010). Our results support the special role of subjective fear in response to trauma-related stimuli as a specific component of PTSD. One hypothesis concerning the difference between anger and fear response and recovery is to attribute fear and anger responses to different underlying neural systems. However, the concept that different brain networks underlie different distinct emotions is controversial (e.g. in favor Ewbank et al., 2009; against

Raz et al., 2016), Another hypothesis focuses more on the differences of the processing of visual stimuli. There is evidence that fear-eliciting stimuli capture more automated attention then angry-eliciting ones (Langeslag & van Strien, 2018). Anger on the other hand might be less the result of an automated process, hence being more influenced by cognitive appraisals processes. Accordingly, there is evidence that maladaptive emotion regulation strategies increase subjective anger following an anger-eliciting stimulus (Fabiansson et al., 2012). Future studies that examine neural and visual processing mechanisms involved will be needed to disentangle the distinct mechanisms affecting fear and anger responses in PTSD.

This study had several limitations. First, although participants were assessed for medication use, they were not assessed for the use of specific psychotropic or somatic (e.g. anti-arrhythmic medication) or any medical treatment, which could have influenced our results, particularly in regard to HRV recordings. However, we did not find differences between the low or high HRV group with regard to the distribution of participants who were and were not taking medication $\chi 2$ (1, N = 69) = 0.96, p = .327 (for details see Table 1). Furthermore, an additional limitation was that the exact date of exposure to each traumatic event was not assessed. Second, the present sample consisted of refugees from a variety of cultural backgrounds. Accredited interpreters experienced in working with mental health-related materials and individuals suffering from mental disorders were used for all assessments. Adapted and validated versions of the applied questionnaires were used whenever available. In the remaining cases English versions were used as a reference for the professional health interpreter, which limited the validity of the assessments. Third, the DSM-5 definition of PTSD was not available at the beginning of the study. Although we adapted the used PTSD definition by including four additional items and excluding one item, which was anticipated to be removed from the DSM-5 definition of PTSD, we did use the DSM-IV wording of several items. Using the exact DSM-5 definition of PTSD might have

resulted in a different prevalence of PTSD diagnosis. Fourth, participants were not excluded if suffering from mental or somatic comorbidities to PTSD (i.e. depression). This limits the attribution of the results exclusively to PTSD, but it reflects the clinical reality in which highly traumatized refugees are unlikely to suffer from PTSD alone (Momartin, Silove, Manicavasagar, & Steel, 2004). Fifth, the experimental setting itself may have been a trauma reminder for some participants, although we put great effort into ensuring that the study setting was not reminiscent of trauma experiences (e.g. only clinical psychologists conducted the experiments). Sixth, due to the complexity of power analysis for mixed models and their interpretation power simulations were not conducted (Arnold, Hogan, Colford, & Hubbard, 2011). Therefore, the power of our study remains unknown. Nevertheless, we did our best to recruit participants in this limited population, making this the study with the biggest sample size of all studies investigating HRV in refugee or post-conflict populations (Liddell et al., 2016; Slewa-Younan et al., 2012; Song et al., 2011).

Findings from this study have potential clinical implications. For example, the finding that low emotion regulation capacity (indexed by low resting HRV) in refugees was linked to diminished anger recovery following exposure to trauma-related stimuli may point to the importance of targeting prolonged anger responses when implementing psychological interventions. Indeed, Nickerson et al. reported that the relationship between trauma exposure and explosive anger in a clinical refugee sample was mediated by reduced impulse control emotion regulation capacities (Nickerson et al., 2015). This is especially important, as maintenance of anger is known to be a predictor of poor treatment outcome (Foa et al., 1995; Forbes, Creamer, Hawthorne, Allen, & Mchugh, 2003; Forbes et al., 2008) and lower social functioning (Hinton et al., 2009).Increasing emotion regulation capacity through emotion regulation skills training (similar to anger and aggression interventions designed for combat veterans with PTSD (Birkley & Schumm, 2016), may therefore be a useful additional

intervention in refugees with anger problems and PTSD. Nevertheless, further research is needed to investigate these interventions and their efficacy in refugee groups. Furthermore, this study highlights that affective reaction to trauma-related cues does not only depend on the evocation of affects, but also on their remission. Therefore, when focusing on a client's affects, the precise assessment of changes in affects over time is important. This stands also true for research investigating affective reaction following trauma cues, with these findings suggesting that it is important to assess not only immediate affective response, but also their recovery.

In conclusion, the aim of this study was to investigate the fear and anger reaction patterns following exposure to trauma-related stimuli and their association with probable PTSD diagnosis and low emotion regulation capacity assessed by HRV at rest. Our findings were twofold. First, probable PTSD diagnosis was associated with increased fear response, which is in line with fear-learning models of PTSD (e.g. Etkin & Wager, 2007). Second, we found that low HRV predicted impaired anger recovery. This is of clinical importance, because increased levels of anger have a disabling role for treatment outcome (Foa et al., 1995) and social functioning (Hinton et al., 2009). However, replication of our results and further research into these complex interactions, also addressing our limitations, is needed.

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Table 1

Group Characteristics for Low Respectively High HRV at Baseline Groups and for Probable PTSD Respectively No PTSD Diagnosis Groups

Variable	Low HRV (n = 38)		High HRV (n = 38)			PTSD (n = 23)		No PTSD (n = 58)		
	Mean	SD	Mean	SD	t	Mean	SD	Mean	SD	t
Age (in years)	35.24	9.29	33.32	9.64	0.89	33.96	7.91	33.95	9.97	-0.01
Men (n)	25		27		0.24a	18		35		1.94a
Duration of Stay (in years)	2.43	3.22	2.70	4.17	-0.31	2.38	2.29	2.56	4.02	0.21
LnHRV at baseline (RMSSD)	2.65	0.51	3.77	0.36	-11.10***	3.16	0.65	3.22	0.74	0.33
Probable PTSD diagnosis (n)	12		9		0.89a					
Medication use (n)	15		10		0.96[a,b]	9		18		0.76[a,c]
Fear at Baseline	1.71	1.06	1.45	0.98	1.12	1.70	0.88	1.52	1.05	-0.72
Anger at Baseline	1.53	1.06	1.26	0.76	1.25	1.57	0.99	1.33	0.87	-1.17

Note. Fear and anger were assessed with a five-point visual scale (min. 1 = not at all, max. 5 = extremely). Trauma Exposure was assessed as the number of different trauma types experienced or witnessed assessed with Harvard Trauma Questionnaire. PTSD Symptoms were assessed by PSSI-Interview. [a]$\chi 2$. [b]$N = 69$. [c]$N = 73$.

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 2

Fear and Anger Reaction Pattern of the Low (n = 38) Respectively High Baseline HRV Group (n = 38) Showing Fixed Effects Estimated with Fully Parameterized Linear Mixed Model for Different Models

		Baseline to Stimuli					Stimuli to Recovery				
DV	Variable	*B*	*SE*	*df*	*t*	*95% CI*	*B*	*SE*	*df*	*t*	*95% CI*
Fear											
	Time	0.63	0.22	117.2	2.83**	[0.19, 1.07]	-0.66	0.17	74.00	-3.20***	[-0.99, -0.32]
	Low HRV	0.05	0.46	74.00	0.12	[-0.86, 0.96]	0.42	0.65	101.9	0.65	[-0.87, 1.71]
	High HRV										
	Time (linear) x Low HRV	0.21	0.32	117.2	0.67	[-0.41, 0.85]	0.03	0.24	74.00	0.11	[-0.45, 0.50]
	Time (linear) x High HRV										
Anger											
	Time	0.92	0.22	114.7	4.24***	[0.49, 1.35]	-0.79	0.16	74.00	-4.83***	[-1.12, -0.46]
	Low HRV	0.24	0.45	74.00	0.53	[-0.65, 1.13]	-0.82	0.64	104.8	-1.28	[-2.08, 0.45]
	High HRV										
	Time (linear) x Low HRV	0.03	0.31	114.7	0.09	[-0.58, 0.64]	0.55	0.23	74.00	2.39*	[0.09, 1.01]
	Time (linear) x High HRV										

Note. DV = Dependent Variable.

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 3

Fear and Anger Reaction Pattern of the Probable PTSD (n = 23) Respectively No PTSD Group (n = 58) Showing Fixed Effects Estimated with Fully Parameterized Linear Mixed Model for Different Models

		Baseline to Stimuli					Stimuli to Recovery				
DV	Variable	*B*	*SE*	*df*	*t*	*95% CI*	*B*	*SE*	*df*	*t*	*95% CI*
Fear											
	Time	1.30	0.28	125.07	4.73***	[0.76, 1.85]	-0.74	0.21	79.00	-3.47**	[-1.16, -0.32]
	Probable PTSD	0.63	0.47	79.00	1.33	[-0.31, 1.57]	-1.25	0.68	106.15	-1.84	[-.2.62, 0.10]
	No PTSD										
	Time (linear) x probable PTSD	-0.80	0.33	125.07	-2.47*	[-1.45, -0.16]	1.36	0.25	79.00	0.54	[-.36, 0.64]
	Time (linear) x No PTSD										
Anger											
	Time	1.30	0.27	122.65	4.78***	[0.76, 1.84]	-0.43	0.22	79.00	-1.98	[-0.87, 0.00]
	Probable PTSD	0.29	0.47	79.00	0.62	[-0.65, 1.23]	-0.50	0.71	107.38	-0.70	[-1.90, 0.90]
	No PTSD										
	Time (linear) x probable PTSD	-0.52	0.32	122.65	-1.64	[-1.17, 0.11]	-0.13	0.26	79.00	-0.52	[-0.65, 0.38]
	Time (linear) x No PTSD										

Note. DV = Dependent Variable.

*$p < .05$. **$p < .01$. ***$p < .001$.

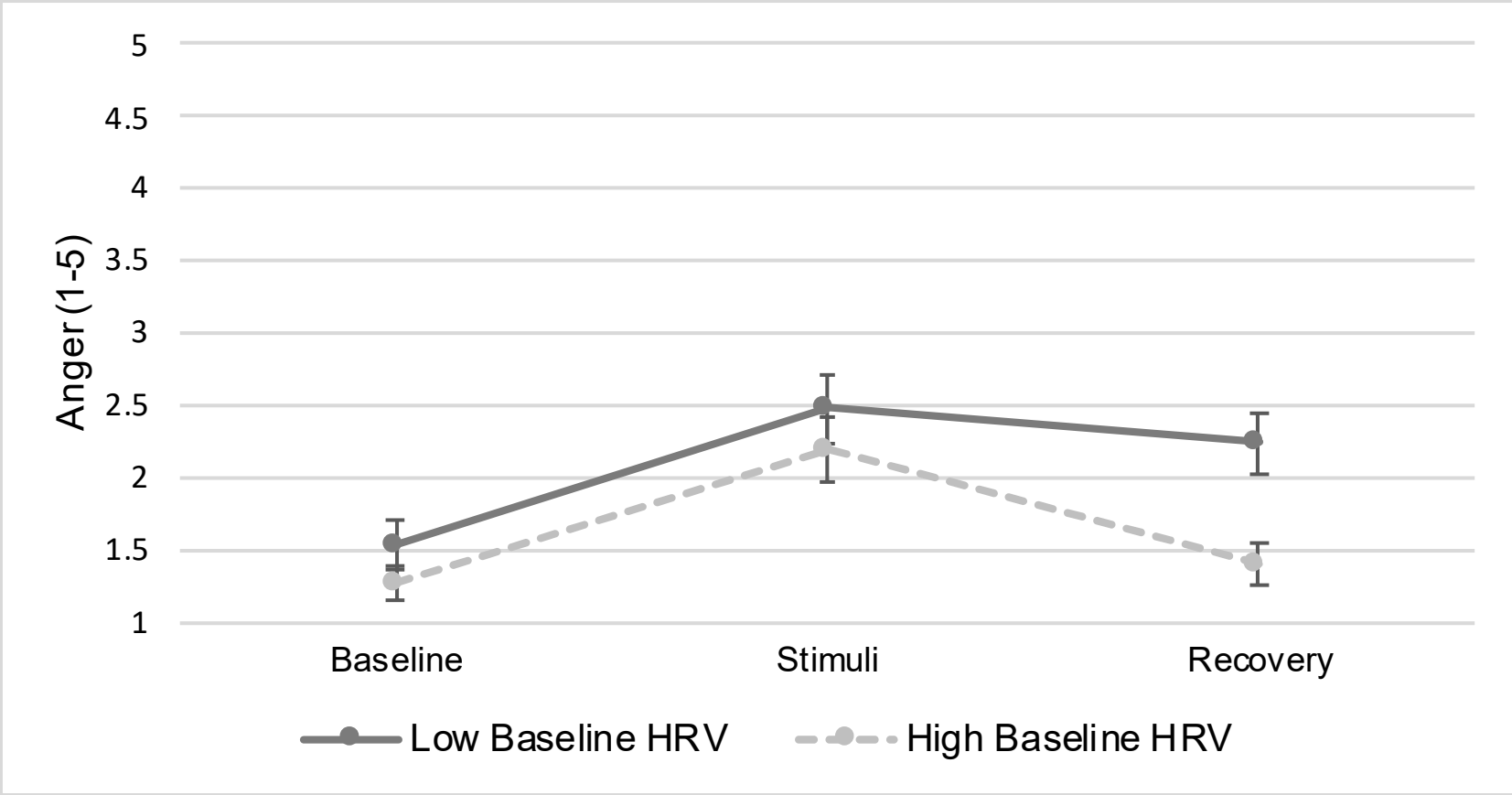

Figure 1. Anger Over Time. Anger reaction pattern of the low (n = 38) respectively high baseline HRV group (n = 38). showing marginal means of anger ratings for each time point. The low baseline HRV group showed an impaired anger recovery. Standard error is presented in the figure by the error bars.

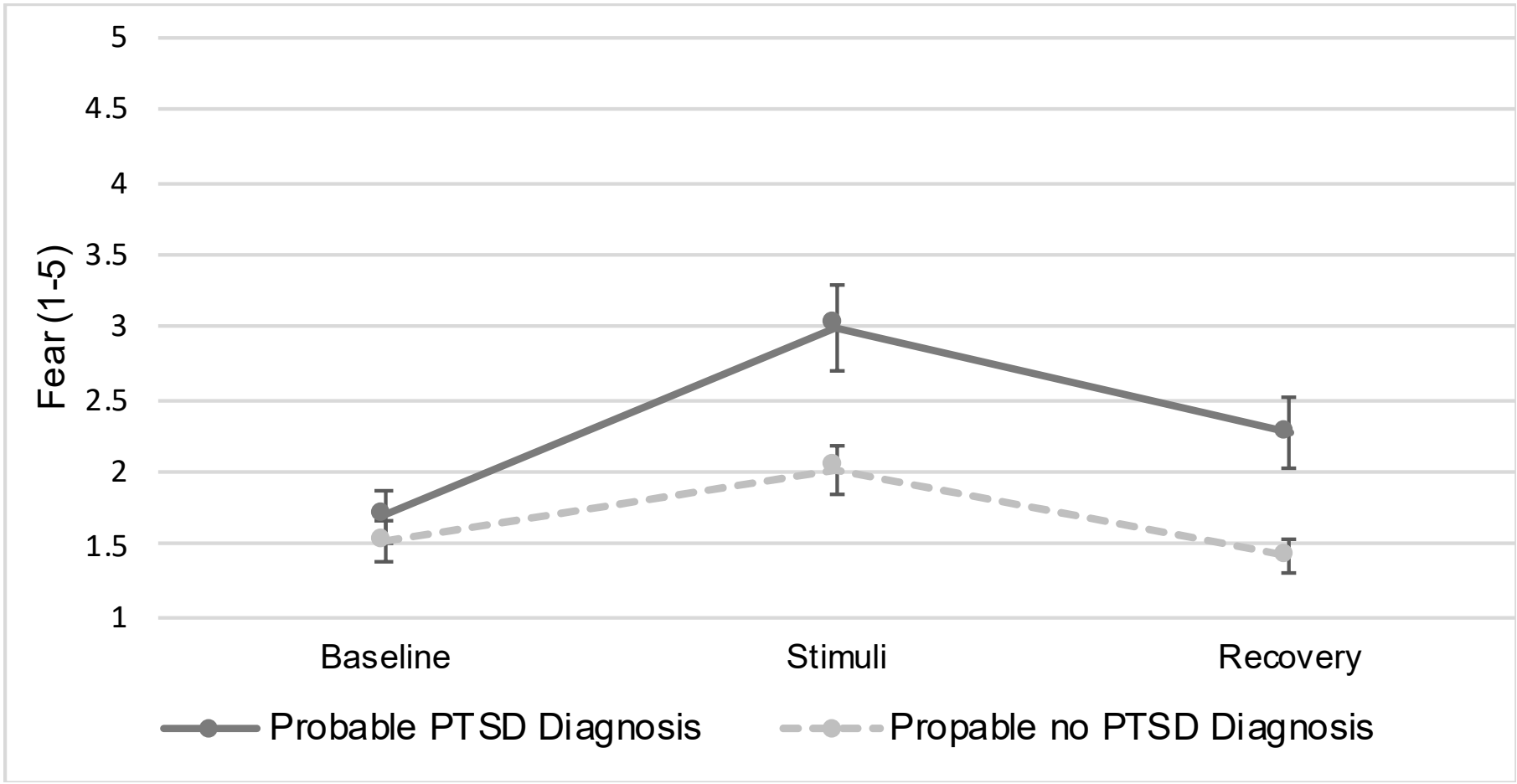

Figure 2. Fear Over Time. Fear reaction pattern of the probable PTSD diagnosis (n = 23) respectively probable no PTSD diagnosis group (n = 58) showing marginal means of fear ratings for each time point. The probable PTSD diagnosis group showed an increased fear response. Standard error is presented in the figure by the error bars.